

LETTERS TO THE EDITOR

- The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.
- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 600 words.
- In general, no letter should contain more than six references (also typed with double spacing).

Is ventricular ectopy a legitimate target for ablation?

SIR,—Symptomatic, benign, ventricular ectopy with a typical left bundle branch block configuration and normal QRS axis without obvious heart disease (normal electrocardiography and echocardiography and no late potentials) is sometimes found by repeated electrocardiography in patients complaining of palpitations.¹ I have seen 14 such cases within the past two years in an outpatient department not specifically designed for arrhythmias.

Most of these patients had had the arrhythmia for many years; symptoms were highly variable and were often associated with stressful periods. Seven of them had traits typical of anxiety disorders² and three of them had had panic attacks which could have explained some of their symptoms (for example, chest pain and dyspnoea). All the patients improved when they were reassured and given benzodiazepines or β blockers.

I wonder whether Gumbrielle's patient really needed four successive antiarrhythmic drugs and finally catheter ablation to treat a benign arrhythmia. It might have been better to treat her with benzodiazepines³ or β blockers in the first instance.

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- 1 Gumbrielle T, Bourke JP, Furniss SS. Is ventricular ectopy a legitimate target for ablation? *Br Heart J* 1994;7:492-4.
- 2 Michels R, Marzuk P. Progress in psychiatry. *N Engl J Med* 1993;329:628-38.
- 3 Shader R, Greenblatt D. Use of benzodiazepines in anxiety disorders. *N Engl J Med* 1993;328:1398-405.

This letter was shown to the authors, who reply as follows:

SIR,—Our patient was a woman with recurrent and remarkably symptomatic unifocal ventricular ectopy. Before medical treatment was started her symptoms did not improve despite repeated reassurance during clinic visits. There was no history, signs, or symptoms to suggest that psychotropic medication would be of benefit. When medical treatment was started, an anti-

arrhythmic agent was chosen for an arrhythmia.

Though we do not wish to recommend widespread ablation of ectopy, we do recognise a small group of patients with troublesome unifocal ectopy arising from the right ventricular outflow tract. As Dr Renard rightly points out, various symptoms that may be provoked by physical or emotional stress occur in the "normal" heart.

The important differential diagnoses are right ventricular dysplasia and idiopathic right ventricular tachycardia, both of which may have a poor prognosis. Of 14 cases with right ventricular outflow tract tachycardia seen over two years, three were found to have right ventricular dysplasia and five displayed identical unifocal ectopy at rest. When this was successfully targeted during radiofrequency ablation, the clinical arrhythmia of sustained or non-sustained ventricular tachycardia was also abolished.

Reassurance and treatment with antiarrhythmic or psychotropic drugs are no longer the best nor the only treatments for this interesting clinical problem

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Increased dispersion of refractoriness in the absence of QT prolongation in patients with mitral valve prolapse and ventricular arrhythmias

SIR,—Tieleman *et al* found that even in the absence of QT prolongation,¹ dispersion of QT and QT_c intervals was greater in patients with mitral valve prolapse and ventricular arrhythmias than in controls. This finding has serious clinical implications.

QT dispersion has been shown to be a marker for sudden cardiac death, ventricular arrhythmia is usually a precursor of sudden cardiac death in mitral valve prolapse, and half of Tieleman *et al*'s patients had either presyncope or syncope. These factors make me wonder whether they found any association between increased QT dispersion and incidence of sudden death in their 220 patients.

Mitral valve prolapse is a common and benign condition except when complications occur. Sudden cardiac death is the most serious, though uncommon, complication. Any marker that can be shown to identify those patients at risk of sudden death will help their management.

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- 1 Tieleman RG, Crijns HJGM, Wiersfeld ACP, Posma J, Hamer HPM, Lie KI. Increased dispersion of refractoriness in the absence of QT prolongation in patients with mitral valve prolapse and ventricular arrhythmias. *Br Heart J* 1995;73:37-40.

This letter was shown to the authors, who reply as follows:

Dr Cheng's question whether we could detect a correlation between increased QT

dispersion and incidence of sudden death in our patients is very interesting and of great importance for the clinical management of patients with mitral valve prolapse.

Priori *et al* reported that patients with long QT syndrome and an increased QT dispersion were more likely to collapse and have a cardiac arrest than comparable patients with normal QT dispersion.¹ Treatment with β blockers or stellate ganglion blockade normalised QT dispersion and relieved symptoms.¹ In patients with heart failure increased QT dispersion was a risk factor for unexpected sudden death, that was independent of left ventricular function.² Because we found increased QT dispersion in patients with mitral valve prolapse and ventricular arrhythmias and because ventricular arrhythmias are the proposed mechanism of sudden death in these patients, Dr Cheng's question is logical. However, it can be answered only by studying a large group of patients with isolated mitral valve prolapse (that is, mitral valve prolapse without other disorders such as ischaemic heart disease, serious aortic valve stenosis, long QT syndrome, or congestive heart failure—all of which can cause sudden cardiac death through ventricular arrhythmias, and some of which are also associated with increased QT dispersion). One hundred and two of our original 220 patients had isolated mitral valve prolapse. Two of 102 died during a median follow up of 7 years (range 2 to 31 years). In neither case was death sudden. One patient died because of acute left heart failure caused by fatal deterioration of mitral regurgitation, the other died from the consequences of multiple myeloma. Because sudden death in patients with isolated mitral valve prolapse is so rare, data from a single hospital cannot answer Dr Cheng's question. Only a central register of cases of sudden death in patients with isolated mitral valve prolapse will establish whether increased QT dispersion is an independent risk factor for sudden death in these patients.

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- 1 Priori SG, Napolitano C, Diehl L, Schwartz PJ. Dispersion of the QT interval. A marker of therapeutic efficacy in the idiopathic long QT syndrome. *Circulation* 1994;89:1681-9.
- 2 Barr CS, Naas A, Freeman M, Lang CC, Struthers AD. QT dispersion and sudden unexpected death in chronic heart failure. *Lancet* 1994;343:327-9.

NOTICE

The 1996 Annual Meeting of the **British Cardiac Society** will take place at the Scottish Exhibition & Conference Centre, Glasgow from 7 to 9 May.